REMARKS

Claims 23-24, 29-34, 36 and 38 are pending after entry of the amendments set forth herein. Claims 1-22, 25-28, 35 and 37 have been canceled without prejudice. Claims 32, 36 and 38 are amended. No new matter is added. Reconsideration is requested.

REJECTIONS UNDER §112, ¶1

Claims 36-37 have been rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement. The Office Action asserts that a disclosure of conjugates of tissue transglutaminase and gluten oligopeptides is not provided in the present application.

Without conceding to the correctness of the rejection, the relevant claims have been amended to recite "tissue transglutaminase linked to SEQ ID NO:12", as set forth in the specification at paragraph 83, "disease indicia include the presence of antibodies specific for the 33-mer of the invention or its deamidated counterparts, glutens, antibodies specific for tissue transglutaminase or tTGase linked to the 33-mer of the invention".

In view of the amendments and remarks, withdrawal of the rejection is requested.

REJECTIONS UNDER §103(A)

Claims 23-24 have been rejected under 35 U.S.C. 103(a) as being unpatentable over Anderson et al. (WO 03104273) in view of Campbell (section 1.3.4. page 29; Monoclonal Antibody Technology (1984) Elsevier Science Publishers).

Applicants respectfully submit that Anderson *et al.* is not available as prior art to the present application. The Anderson *et al.* application was filed on June 5, 2003 and published December 18, 2003, both after Applicants' priority date of November 20, 2002.

Applicants note that a US counterpart to the Anderson *et al.* application has been published in the United States as Application no. 20060178299. This application claims priority to Great Britain filing 01212885.8, filed June 5, 2002. However, Applicants respectfully submit that the published US application is not prior art to the present application, as it is not available under 35 U.S.C. 102(e).

As set forth in 35 U.S.C. 102(e), the invention must be described in "an application for patent, published under section 122(b), by another filed in the United States before the invention by the applicant for patent". Applicants submit that the priority application filed in Great Britain

does not provide for an application "filed in the United States", as required by the law. Therefore, Anderson *et al.* is only effective as a reference as of its international filing date, June 5, 2003, and is not earlier than Applicants' priority date.

In view of the above remarks, withdrawal of the rejection is requested.

Claims 23-24 have been rejected under 35 U.S.C. 103(a) as being unpatentable over Arentz-Hansen *et al.* in view of Campbell.

The Office Action states that Arentz-Hansen *et al.* study several alpha-gliadins for the CD412/CD387 recognition. Arentz-Hansen *et al.* reports that one particular peptide, alpha 2 (62-75) PQPQLPYPQPQLPY, has particular function to stimulate T cell recognition (See Table 11; page 606). Such 14-mer peptide is encompassed within SEQ ID NO. 12. It is further submitted in the Office Action that it would have been obvious to produce antibody when the antigen is identified and characterized.

Applicants respectfully submit that Arentz-Hansen et al. does not teach or suggest the presently claimed invention. Applicants' claims are directed to an antibody that specifically binds to a purified oligopeptide having the amino acid sequence LQLQPFPQPQLPYPQPQLPYPQPQLPYPQPQPF (SEQ ID NO:12).

While the peptides disclosed by Arentz-Hansen have similarities to those set forth by Applicants, the actual antigenic peptide in the publication differs from that of the present claims. The term "epitope" as used by Arentz-Hansen refers to an epitope that is recognized by a T cell. While there can be considerable overlap between epitopes recognized by antibodies and epitopes recognized by T cells, there are also significant differences. Many of these differences are a reflection of the requirement that a peptide be "presented" by an HLA antigen in order to be recognized by a T cell.

As stated in the abstract of the reference:

> The great majority of patients that are intolerant of wheat gluten protein due to celiac disease (CD) are human histocompatibility leukocyte antigen (HLA)-DQ2^{+}, and the remaining few normally express HLA-DQ8. These two class II molecules are chiefly responsible for the presentation of gluten peptides to the gluten-specific T cells that are found only in the gut of CD patients but not of controls. Interestingly, tissue transglutaminase (tTG)-mediated deamidation of gliadin plays an important role in recognition of this food antigen by intestinal T cells. Here we have used recombinant antigens to demonstrate that the intestinal T cell response to α-gliadin in adult CD is focused on two immunodominant, DQ2-restricted peptides that overlap by a seven-residue fragment of gliadin. We show that tTG converts a glutamine residue within this fragment into glutamic acid and that this process is critical for T cell recognition. (underlining added)

Thus, it is not the native peptide, which sequence is a subset of SEQ ID NO:12, that is an epitope in the prior art, but a deamidated version of the peptide. As shown in Figure 2 of Arentz-Hansen, it is only after deamidation that the peptides act as epitopes for the T cell clones.

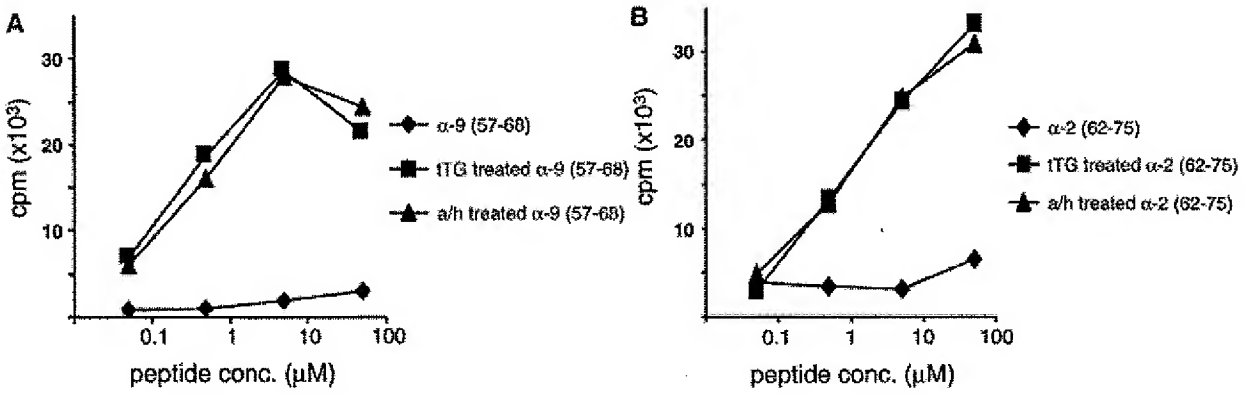

Figure 2. Recognition of peptides α-9(57–68) and α-2(62–75) by two TCCs. Peptides α-9(57–68) (A) and α-2(62–75) (B) were tested in their native state (♦) or after treatment with human tTG (■) or acid/heat (a/h; ▲) for their ability to induce proliferation in (A) TCC CD387 E34 and (B) TCC CD412 R5.32. Responses are given in cpm.

Applicants respectfully submit that Arentz-Hansen teaches away from the present invention, and teaches that only a deamidated derivative of the peptide disclosed therein would be an effective epitope for T cell immune recognition of the peptide disclosed therein. While reserving the right to pursue claims of the original scope in a later filing, in order to expedite prosecution Applicants have canceled the claims or portions of claims herein that reference such deamidated epitopes.

Once of skill in the art would not be led to produce an antibody directed at the peptide set forth in the present claims, without the guidance of the present application as to the importance of SEQ ID NO:12 in the development of Celiac Sprue.

In view of the above amendments and remarks, withdrawal of the rejection is requested.

Claims 23-24, 29-35, and 38 have been rejected under 35 U.S.C. 103(a) as being unpatentable over Hausch et al. (US 7,303,871) in view of Campbell. The Office Action states that Hausch *et al.* disclose a glutenase resistance peptide SEQ ID No. 12, and further notes that the patent claims priority to provisional application no. 60/380,761.

Applicants respectfully submit that Hausch *et al.* is not available as art for purposes of 35 U.S.C. 103 against the present application. A Declaration by the present inventors is attached

herewith, providing for the attribution of subject matter disclosed and unclaimed in US 7,303,871.

In view of the above remarks and attached Declaration, withdrawal of the rejection is requested.

CONCLUSION

Applicant submits that all of the claims are in condition for allowance, which action is requested. If the Examiner finds that a telephone conference would expedite the prosecution of this application, please telephone the undersigned at the number provided.

The Commissioner is hereby authorized to charge any underpayment of fees associated with this communication, including any necessary fees for extensions of time, or credit any overpayment to Deposit Account No. 50-0815, order number STAN-258US5.

Respectfully submitted,
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Date: July 14, 2009

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